Mitral Valve Replacement Early After Myocardial Infarction: Attendant High Risk of Left Ventricular Rupture

JOHN G. HAROLD, MD, FACC, TIMOTHY M. BATEMAN, MD, FACC, LAWRENCE S. C. CZER, MD, FACC, AURELIO CHAUX, MD, FACC, JACK M. MATLOFF, MD, FACC, RICHARD J. GRAY, MD, FACC

Los Angeles, California

Between 1969 and 1983, 608 patients underwent mitral valve replacement surgery at Cedars-Sinai Medical Center. Perioperative rupture of the left ventricular myocardium complicated seven operations (1.2%), five of them in the 247 patients with concomitant ischemic heart disease. Six ruptures were fatal. Relative incidences of seven previously hypothesized predisposing factors were determined for patients with and without myocardial rupture. In addition, because of the apparent frequency of association with ischemic heart disease and because all ruptures were posterior or posterolateral, patients were also categorized by prior history of posterior myocardial infarction: 177 patients had none, whereas 49 patients had a remote and 21 patients a recent (≤1 month) posterior wall infarct. Four ruptures (accounting for 57% of all ruptures) occurred in the 21 patients (19% incidence) with a recent posterior infarct, compared with only three ruptures in the 587 patients (0.5%) without a recent posterior wall infarct (p = 0.000).

None of the factors of age, sex, valve pathology, etiology of valve lesion, concomitant coronary disease, valve substitute or intraoperative myocardial preservation were associated with perioperative rupture. These data establish a low overall incidence of ventricular rupture after mitral valve replacement, high fatality and possible etiologic association with recent posterior wall infarction.

(J Am Coll Cardiol 1987;9:277–82)

Mitral valve replacement has a high mortality rate when there is concomitant coronary artery disease (1,2). Among the causes, left ventricular myocardial rupture is reported to be rare (0.2 to 2%) but is frequently fatal (3–10). Previous studies (11–18) have suggested an association with a small left ventricular cavity, ventriculovalvular disproportion, surgical injury to the myocardium and sudden overdistension of the left ventricle after mitral valve replacement (with or without interruption of papillary muscle-valve continuity). The importance of ischemic heart disease has never been examined. Observation of a fatal posterior wall rupture early after mitral valve replacement in a patient with a recent posterior infarct prompted review of our entire operative experience with this complication.

From the Division of Cardiology and the Department of Thoracic and Cardiovascular Surgery, Cedars-Sinai Medical Center, and the Department of Medicine, UCLA School of Medicine, Los Angeles, California. This study was presented in part at the 34th Annual Scientific Sessions of the American College of Cardiology, Anaheim, California, March 1984.

Manuscript received June 25, 1986; revised manuscript received August 20, 1986, accepted September 5, 1986.

Address for reprints: Timothy M. Bateman, MD, Cedars-Sinai Medical Center, Room 6215, 8700 Beverly Boulevard, Los Angeles, California 90048.

Methods

Study patients. Between 1969 and 1983, 608 patients underwent mitral valve replacement surgery at Cedars-Sinai Medical Center. Their mean age was 60 ± 12 years (mean ± SD) (range 18 to 86), and 255 (42%) were male. Forty-three percent had isolated mitral valve replacement, 32% had both valve replacement and coronary artery bypass grafting, 19% had double valve replacement and 6% had double valve replacement and bypass surgery. The valve substitutes included 285 porcine, 167 Harken, 154 St. Jude and 2 Björk-Shiley valve prostheses.

The etiology of the mitral valve disease was rheumatic in 328 patients (54%), ischemic in 158 patients (26%) and secondary to other causes in 122 patients (20%). The secondary causes included mitral anular dilation associated with left ventricular dilation, mitral valve prolapse, infective endocarditis, Marfan's syndrome, congenital mitral regurgitation and degenerative disease of the mitral valve.

Operative procedure. Mitral valve replacement was performed under general endotracheal anesthesia with hemodynamic monitoring (radial artery and pulmonary artery thermodilution catheters) and cardiopulmonary bypass

0735-1097/87/$3.50

using a bubble oxygenator. Between 1969 and 1978, 284 patients had intermittent ischemic cardiac arrest (aortic cross clamping) for myocardial preservation. In the subsequent 324 patients, moderate systemic hypothermia (20°C to 32°C) and intermittent multidose potassium cardioplegia with either a crystalloid (St. Thomas solution) or modified blood solution at 4°C infused into the aortic root (or coronary ostia) were used. If concomitant coronary artery bypass was performed, it was accomplished utilizing autologous reversed saphenous vein or internal mammary artery, or both. Distal bypass anastomoses were performed first, followed by mitral valve excision and replacement and finally the proximal anastomoses.

Clinical data. The Cedars-Sinai cardiac surgery computerized data base prospectively enters more than 100 variables on all patients undergoing open-heart surgery, who are then followed up at 1 year intervals by telephone and written questionnaire. This data base provided preoperative and early postoperative historic, electrocardiographic, coronary angiographic and ventriculographic information and demographic and late follow-up information.

Preoperative variables were age, sex, symptoms (heart failure, angina, arrhythmias); mitral valve lesion (mitral stenosis, mitral regurgitation or mixed); origin of the mitral valve disease (rheumatic, ischemic or other causes as outlined under Methods); and the presence of coronary artery disease or recent (≤1 month) or remote infarction.

Cardiac catheterization was performed in all patients before the operative procedure. Coronary artery disease was defined as 50% or greater luminal diameter narrowing in at least one coronary artery.

Transmural myocardial infarction was diagnosed by clinical history and electrocardiographic findings (Q waves), radionuclide blood pool (akinetic wall motion) or thallium-201 redistribution (irreversible perfusion defect) scintigrams or left ventricular angiography (akinesia).

Specific criteria for posterior myocardial infarction were at least one of the following: 1) electrocardiographic: an acute increase in the height and width of the R wave in precordial leads V_1 and V_2 with or without associated inferior or lateral wall, or both, pathologic Q waves or ST-T wave change; 2) *scintigraphic:* posterolateral or posterobasal akinesia on radionuclide ventriculography or perfusion defect nonreversibility on rest or exercise thallium-201 redistribution scintigraphy; 3) *cineangiographic:* posterior wall akinesia; 4) *surgical:* posterior epicardial scarring; and 5) *pathologic:* gross and histologic documentation of posterior infarction at postmortem examination.

Data analysis. Because past studies suggested a possible association with them, seven variables were examined in this study to evaluate their relation to perioperative myocardial rupture. The relative incidence of rupture by sex, age (> versus ≤ 60 years), predominant valve lesion, cause of the valve lesion, coronary artery disease, form of myocardial protection and type of valve substitute was tested for statistical significance using Fisher's exact test.

After preliminary analysis of the data, patients were classified into four groups according to mitral valve replacement: 1) without coronary artery disease (Group 1, 361 patients), 2) with coronary artery disease but no previous posterior myocardial infarction (Group 2, 177 patients), 3) with coronary artery disease and remote posterior myocardial infarction (Group 3, 49 patients), and 4) with coronary artery disease and recent posterior myocardial infarction (Group 4, 21 patients).

Results

Seven left ventricular free wall ruptures occurred during this 14 year experience with 608 mitral valve replacements giving a 1.2% overall incidence; 6 of these were fatal. All free wall ruptures involved the true posterior or posterolateral wall and were between the atrioventricular groove and the posteromedial papillary muscle remnant (Fig. 1). They occurred on postoperative day 0 (four patients), 4, 5 and 36, respectively. In the one surviving patient (Patient 3, Table 3), the rupture was recognized intraoperatively and was successfully repaired.

Table 1 shows the relative incidence of posterior wall rupture in the four patient groups categorized by the presence of coronary artery disease and history of posterior wall myocardial infarction. Fifty-seven percent of all ruptures (four of seven) were in the 21 patients with a recent posterior myocardial infarction. By comparison, rupture occurred in 0 to 0.6% of the patients in each of the other groups. During this same period there were four ruptures involving the atrioventricular groove. None of these had associated recent or remote posterior myocardial infarction.

Clinical associations. Table 2 demonstrates that recent posterior myocardial infarction was the only tested variable

Figure 1. Diagrammatic representation of the site of posterior wall rupture in this patient series. All seven cases of rupture were between the atrioventricular groove and the posteromedial papillary muscle remnant. LA = left atrium; LV = left ventricle.

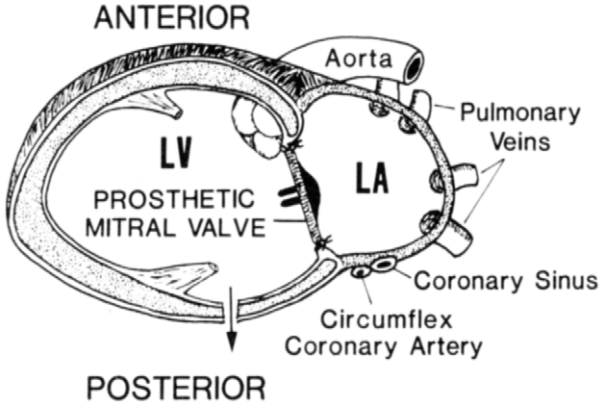

Table 1. Relative Incidence of Posterior Wall Rupture in Four Groups of Patients With Mitral Valve Replacement

Group	No. of Patients	No. of Ruptures
1	361	2 (0.6%)
2	177	1 (0.6%)
3	49	0
4	21	4 (19%)
Total	608	7 (1.2%)

Definition of groups with mitral valve replacement: 1 = no coronary artery disease; 2 = coronary artery disease without previous posterior infarction; 3 = coronary artery disease and remote posterior infarction; and 4 = coronary artery disease and recent posterior infarction.

that was statistically more frequent in patients with perioperative rupture. Compared with a 0.7% incidence of rupture with other valve prostheses, there was a 2.4% rupture rate with the Harken valve; this difference was of borderline significance ($p = 0.09$).

Table 3 summarizes the relevant data for the seven patients with free posterior wall rupture complicating their valve replacement. These patients were elderly (average age 69 years), and five were female; the majority had mitral stenosis. Two patients had combined aortic and mitral valve replacement. Five patients had coronary artery disease and four of these had posterior myocardial infarction within 1 month of surgery. The four Harken valves were of medium size (No. 4), as were the porcine (No. 29) and two St. Jude (No. 29, No. 31) valve prostheses.

Table 4 examines the 21 patients in whom mitral valve replacement was performed within 1 month of posterior myocardial infarction. No statistical comparison was performed because of small numbers, but there were no overwhelming features that distinguished the 17 patients without rupture from the 4 patients with this complication.

Careful postmortem examination of five of the ruptured hearts was performed. In addition, the one intraoperative rupture was assessed visually by the attendant surgeons as to cause. In no case was there evidence that rupture was due to direct surgical injury of the myocardium.

Illustrative Cases (Table 3)

Case 1. A 73 year old woman with coronary artery disease and mitral stenosis had emergent mitral valve replacement in the setting of acute inferoposterior myocardial infarction. Nine hours after surgery, the patient had a massive hemorrhage from her chest tubes. At autopsy, the pros-

Table 2. Impact of Risk Factors on Incidence of Posterior Ventricular Wall Rupture After Mitral Valve Replacement

Variable	Patients at Risk (n = 608)	Posterior Wall Rupture		p Value
		No.	%	
Age				
<60 years	219 (36%)	1	0.5	NS
60 or older	389 (64%)	6	1.5	
Sex				
Male	255 (42%)	2	0.8	NS
Female	353 (58%)	5	1.4	
Mitral lesion				
Stenosis	85 (14%)	3	3.5	NS
Mixed or predominant regurgitation	523 (86%)	4	0.8	
Coronary disease				
Yes	292 (48%)	5	1.7	NS
No	316 (52%)	2	0.6	
Recent posterior infarct				
Yes	21 (3.5%)	4	19	0.0
No	587 (96.5%)	3	0.5	
Myocardial arrest				
Anoxic	284 (47%)	5	1.8	NS
Cardioplegic	324 (53%)	2	0.6	
Valve substitute				
Harken	167 (27%)	4	2.4	NS
Porcine	285 (47%)	1	0.4	
St. Jude	154 (25%)	2	1.3	
Björk-Shiley	2 (0.3)	0	0	

Table 3. Seven Cases of Posterior Wall Rupture Complicating Mitral Valve Replacement

Patient	Age (yr) & Sex	Lesion	Operation	Mitral Prosthesis	CAD	Recent PMI
1	73F	MS	MVR-ICC	Harken	Yes	Yes
2	72F	MS	MVR-ICC	Harken	No	No
3*	55M	MS	MVR-ICC	Harken	Yes	No
4	68F	MS/AS	DVR-ICC	Harken	No	No
5	74M	MR/AI	DVR-ICC	Porcine	Yes	Yes
6	67F	MR	MVR-HCP	St. Jude	Yes	Yes
7	75F	MS	MVR-HCP	St. Jude	Yes	Yes

*Rupture recognized and repaired intraoperatively, and the patient survived. All other patients died as a result of the myocardial rupture. AI = aortic insufficiency; AS = aortic stenosis; CAD = coronary artery disease; DVR = double valve replacement; HCP = hypothermic cardioplegia; ICC = intermittent cross clamping; MR = mitral regurgitation; MS = mitral stenosis; MVR = mitral valve replacement; PMI = posterior myocardial infarction.

thetic valve (Harken) was found to have eroded through the recently infarcted posterior wall.

Case 5. A 74 year old white man with mitral and aortic insufficiency underwent a lengthy and technically difficult double valve replacement (Hancock prostheses). On the fourth postoperative day, the patient hemorrhaged through the mediastinal chest tubes and could not be resuscitated. At autopsy there were massive left and right ventricular hypertrophy, recent necrosis of the posterior and lateral left ventricular walls and perforation of the infarcted ventricle at the site of contact with one of the commissural valve posts.

Case 6. A 67 year old woman with mitral regurgitation and coronary disease had mitral valve replacement with a St. Jude prosthesis 2 weeks after posterobasal myocardial infarction. Seven hours postoperatively, the patient had a sudden rise in systolic blood pressure, followed by exsanguinating hemorrhage through the mediastinal drainage tubes.

Table 4. Mitral Valve Replacement in 21 Patients Early After Posterior Infarction: Comparison of Patients With and Without Rupture

	Rupture (n = 4)	No Rupture (n = 17)
Age (yr)	72	65
Female sex	75%	29%
Predominant lesion		
MS	2 (50%)	3 (18%)
MR or mixed	2 (50%)	14 (82%)
MVR	3 (75%)	16 (94%)
DVR	1 (25%)	1 (6%)
Aortic cross clamping	2 (50%)	6 (35%)
Cold cardioplegia	2 (50%)	11 (65%)
Prosthesis		
Harken	1 (25%)	3 (18%)
Porcine	1 (25%)	8 (47%)
St. Jude	2 (50%)	6 (35%)

Abbreviations as in Table 3.

At autopsy, an oval-shaped rupture of the free posterolateral wall was noted in a region characterized by thinning and histopathologic changes of a 2 week old myocardial infarction.

Case 7. A 75 year old white woman with mitral regurgitation had electrocardiographic evidence of acute posterolateral infarction immediately after mitral valve replacement (St. Jude prosthesis). On the fourth postoperative day, the patient had a terminal cardiopulmonary arrest. Autopsy revealed that the proximal left circumflex artery was encircled and partially occluded by an atriotomy suture. There was a large necrotizing posterolateral infarction in the distribution of the ligated vessel with associated rupture and hemopericardium.

Discussion

Ventricular rupture complicating mitral valve replacement. Left ventricular rupture after mitral valve replacement has been associated with a variety of possible predisposing causes. This is the first report to indicate the likely importance of recent posterior wall infarction as a risk factor. In our series of 608 mitral valve replacements, there were 7 cases complicated by ventricular rupture, 4 of which were in patients with existent regions of myocardial necrosis involving the true posterior or posterolateral wall. Six of the seven ruptures were fatal. These data establish a low overall incidence of ventricular rupture after mitral valve replacement (1.2%), an etiologic association with recent posterior infarction (in our series 57% of all ruptures were in such patients) and a high fatality rate (86%). Posterior infarctions older than 1 month were not associated with an increased risk for cardiac rupture. Recognition of this increased risk is potentially important in view of the recent trends for acute revascularization and valve replacement after myocardial infarction. Our patient population is char-

acteristic of recent experience with mitral valve surgery, in that 41% of patients had coexisting coronary artery disease.

Possible etiologic mechanisms of rupture. Extensive published data address an association between acute myocardial infarction and cardiac rupture. In 1647 Harvey (19) described a patient with free left ventricular wall rupture who died after an episode of prolonged chest pain. Today, cardiac rupture complicates up to 8% of cases of acute infarction and contributes substantially to mortality (20). A higher incidence has been reported in women and the elderly; the peak incidence is in those over 70 years of age (21). There is also a correlation between hypertension in the acute phase of myocardial infarction and cardiac rupture (22).

The review of Rasmussen et al. (20) of 72 consecutive cases of infarction-related cardiac rupture (20) may provide insight into why acute rupture may be more common in the setting of fresh myocardial necrosis and valve replacement. In their series, the peak incidence of rupture was between the second and eighth days after infarction, coinciding with the period in which myocardial necrosis and softening are maximal. As the most common site of rupture was the junction of infarcted with healthy tissue, the increased tensile strength generated at this site is a probable pathogenic factor.

It has been reported (23) that the rigid prosthetic valve ring exerts increased shear forces across the ventricular myocardium, which may be most important posteriorly where the ventricular myocardium is thinnest. These high shear forces may further increase the likelihood of cardiac rupture if the posterior wall has been freshly infarcted. Similar to the reported experience of others, we found that advanced age, female sex and presence of mitral stenosis appeared to increase the risk of this complication in the high risk group. However, none of these was an independent risk factor. Most ruptures occurred with medium-sized (the most common size mitral valve used at our center) (24) low profile valve prostheses, making it unlikely that valve size and attendant mechanical impingement on the myocardium were primary inciting factors for rupture.

Possible treatment strategies. Although no study provides guidance, therapeutic options to avoid cardiac rupture that would seem reasonable are careful control of postoperative blood pressure and ventricular volume in those high risk patients with mitral valve replacement performed in the setting of recent posterior infarction. The maintenance of a high index of suspicion is also important so that appropriate emergent intervention can be performed if required. Mitral valve replacement in the setting of recent posterior myocardial infarction should be delayed as long as possible so that complete healing of the posterior wall can occur.

Conclusions. Our experience confirms that free posterior wall rupture after mitral valve replacement is usually fatal. The results of this study suggest that patients with coexisting coronary artery disease and recent posterior wall infarction present an increased risk for cardiac rupture after mitral valve replacement. This increased risk is important in view of the increasing number of patients undergoing acute revascularization and mitral valve replacement after acute myocardial infarction.

References

1. Czer LS, Gray RJ, DeRobertis MA, et al. Mitral valve replacement: impact of coronary artery disease and determinants of prognosis after revascularization. Circulation 1984;70(suppl I):I-198–207.
2. Czer L, Matloff J, Gray R, Bateman TM, Chaux A. Mitral valve replacement with coronary artery disease. In: Duran C, Angell WW, Johnson AD, Oury JH, eds. Recent Progress in Mitral Valve Disease. London: Butterworth, 1984:304–19.
3. Björk VO, Henze A, Rodriguez L. Left ventricular rupture as a complication of mitral valve replacement: surgical experience with eight cases and a review of the literature. J Thorac Cardiovasc Surg 1977;73:14–20.
4. Treasure RL, Rainer WG, Strevey TE, Sadler TR. Intraoperative left ventricular rupture associated with mitral valve replacement. Chest 1974;66:511–4.
5. Zacharias A, Groves LK, Cheanvechai C, Loop FD, Effler DB. Rupture of the posterior wall of the left ventricle after mitral valve replacement. J Thorac Cardiovasc Surg 1975;69:259–63.
6. Chi S, Beshore R, Gonzales-Lavin L. Left ventricular wall rupture after mitral valve replacement: report of successful repair in 2 patients. Ann Thorac Surg 1976;22:380–2.
7. Wolpowitz A, Barnard MS, Sanches HE, Barnard CN. Intraoperative posterior left ventricular wall rupture associated with mitral valve replacement. Ann Thorac Surg 1978;25:551–4.
8. Nunez L, Gil-Aguado M, Cerron M, Celemin P. Delayed rupture of the left ventricle after mitral valve replacement with a bioprosthesis. Ann Thorac Surg 1979;27:465–7.
9. Bortolotti U, Thiene G, Casarotto D, Mazzucco A, Galluci V. Left ventricular rupture following mitral valve replacement with a bioprosthesis. Chest 1980;77:235–7.
10. Gosalbez F, de Linera FA, Cofino JL, Naya JL, Rodriguez J, Ortuna A. Isolated mitral valve replacement and ventricular rupture: presentation of 6 patients. Ann Thorac Surg 1980;31:105–10.
11. Roberts WC, Morrow AG. Causes of early postoperative death following cardiac valve replacement: clinicopathologic correlations in 64 patients studied at necropsy. J Thorac Cardiovasc Surg 1967;54:422–37.
12. Roberts WC. Complications of cardiac valve replacement: characteristic abnormalities of prostheses pertaining to any or specific site. Am Heart J 1982;103:113–22.
13. Nili M, Salomon J, Halevi A, Schuchman E, Levy MJ. Left ventricular rupture after mitral valve replacement. Report of two cases and a review of the literature. Scand J Thorac Cardiovasc Surg 1981;15:235–8.
14. MacVaugh H, Joyner CR, Johnson J. Unusual complications during mitral valve replacement in the presence of calcification of the annulus. Ann Thorac Surg 1971;11:336–42.
15. Cobbs BW, Hatcher CR, Craver JM, Jones EL, Sewell CW. Transverse midventricular disruption after mitral valve replacement. Am Heart J 1980;99:33–50.
16. Roberts WC, Isner JM, Virmani R. Left ventricular incision midway between the mitral annulus and the stumps of the papillary muscles during mitral valve excision with or without rupture or aneurysmal formation: analysis of 10 necropsy patients. Am Heart J 1982; 104:1278–87.

17. Wild LM, Lajos TZ, Lee AB, Wright J. Left ventricular laceration due to a stented prosthesis. Chest 1980;77:216–7.

18. Isner JM. Case Records of the Massachusetts General Hospital. N Engl J Med 1983;309:1233–42.

19. Harvey W. Complete Works (Willis R, translator). London: Sydenham Society, 1847:127 (New York: Johnson Reprint Corporation, 1965).

20. Rasmussen S, Leth A, Kjoller E, Pedersen A. Cardiac rupture in acute myocardial infarction: a review of 72 consecutive cases. Acta Med Scand 1979;205:11–6.

21. Bates RJ, Beutler S, Resnekov L, Anagnastopoulous CE. Cardiac rupture—challenge in diagnosis and management. Am J Cardiol 1977;40:429–37.

22. Ross RM, Young JA. Clinical and necropsy findings in rupture of the myocardium. A review of forty-three cases. Scott Med J 1963;8:222–6.

23. Huikuri H. Effect of mitral valve replacement on left ventricular function in mitral regurgitation. Br Heart J 1983;49:328–33.

24. Czer LSC, Matloff JM, Chaux A, et al. Comparative clinical experience with porcine bioprosthetic and St. Jude valve replacement. Chest (in press).